Public Health Impact of Coal and Electricity Consumption

Risk–Benefit Balance Varies by Country

Access to electricity contributes to good health by powering infrastructure for clean drinking water and sanitation and by reducing the need for indoor burning of coal, wood, and other solid fuels. But these benefits can be offset by health threats posed by the emissions from fossil fuel–based electricity production—direct public health effects attributable to particulate matter, sulfur and nitrous oxides, volatile organic compounds, carbon monoxide, and ozone are estimated to account for more than 70% of the external costs of power generation (i.e., costs not factored into the price paid for electricity). A multitiered analysis of the relationship between coal consumption, electricity use, and health outcomes uses three complementary data sets to compare positive and negative health effects of power generation [***EHP* 119(6):821–826; Gohlke et al.**].

The authors developed an autoregressive time-series model of infant mortality, life expectancy, electricity consumption, and coal consumption for 41 different countries over the period 1965–2005. They divided the countries into three groups depending on infant mortality and life expectancy rates as of 1965: countries with high infant mortality and low life expectancy, those with moderately high infant mortality and medium to high life expectancy, and those with low infant mortality and high life expectancy.

Model predictions suggested infant mortality would decrease with increasing electricity consumption over time, but only in countries that started off with high infant mortality and low life expectancy, a group that included Algeria, Brazil, India, Indonesia, Pakistan, Peru, South Africa, and Turkey. Models did not predict a change in life expectancy with increased electricity use, but did predict a decrease in life expectancy with increased coal consumption in countries with moderate infant mortality and life expectancy in 1965. In addition, infant mortality was predicted to increase with increased coal consumption in those countries with low infant mortality and high life expectancy.

The authors compared these results with estimates from two independent methods for modeling health effects of energy-related environmental exposures. The first method, the World Health Organization's Environmental Burden of Disease model, estimates the burden of human disease related to outdoor air pollution, indoor air pollution, drinking water, and sanitation. The second method, the Greenhouse Gas and Air Pollution Interactions and Synergies model developed by the International Institute for Applied Systems Analysis, estimates potential life-shortening effects of pollutant emissions from coal-fired power plants. Estimates from both models were consistent with those derived from the authors' autoregressive model.

The study's limitations include a lack of comprehensive data for variables such as education level, vaccination rates, and health care access and/or expenditures. However, the consistency of the results of the three analyses strongly supports the authors' conclusions and highlights ways that human health impacts might be integrated into climate change mitigation and energy policy research.

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Outside the coal-fired Guru Hargobind Thermal Power Station, Bathinda, Punjab, India

Chemical Count

Quantifying Exposures in Pregnant Women

A nationally representative assessment of pregnant women's exposure to 163 chemicals reveals what the authors term "ubiquitous exposure to multiple chemicals during a sensitive period of development" [***EHP* 119(6):878–885; Woodruff et al.**]. The new study is based on samples collected and analyzed as part of the National Health and Nutrition Examination Survey (NHANES) 2003–2004.

The researchers assessed data for 268 pregnant women between the ages of 15 and 44. Chemical analytes assessed included metals, perfluorinated compounds, organochlorine pesticides, organophosphate insecticide metabolites, phthalates, polybrominated diphenyl ethers (PBDEs), polycyclic aromatic hydrocarbons (PAHs), phenols, polychlorinated biphenyls (PCBs), dioxin-like chemicals, perchlorate, triclosan, and volatile organic compounds. Not all analytes were measured in all women.

The study showed the pregnant women had widespread exposure to substances banned decades ago as well as contemporary contaminants. Several of the chemical analytes assessed were detected in 99–100% of the pregnant women. There was substantial variation in the levels of individual analytes to which pregnant women were exposed. Most notably, the difference between the geometric mean and 95th percentile for phthalates and one PBDE, BDE-153, varied by more than an order of magnitude. More research is needed to identify the major sources of exposure to these compounds among pregnant women and the general population, the authors say.

Although no health effects were assessed as part of this study, levels of many chemicals detected—including mercury, phthalates, PBDEs, and PCBs—were similar to those associated with adverse reproductive and developmental effects in epidemiologic studies. The study also showed that many women were exposed to multiple chemicals that may contribute to the same adverse outcomes. For example, perchlorate, PCBs, PBDEs, and triclosan have all been associated with changes in maternal thyroid hormones, whereas mercury, lead, and PCBs can all harm the developing brain.

The authors point out that exposure to multiple chemicals that act on the same adverse outcome can have a greater effect than exposure to an individual chemical. The National Academy of Sciences recommends accounting for multiple exposures, as well as exposures that occur during sensitive periods of development, in order to improve assessment of chemical risks across the U.S. population.

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Rotenone and Paraquat Linked to Parkinson's Disease

Human Exposure Study Supports Years of Animal Studies

A growing body of evidence suggests pesticides may play a role in Parkinson's disease (PD) in humans. Self-reported PD has been associated with lifetime use of pesticides, and animal studies have suggested that the pesticides paraquat and rotenone can cause oxidative stress and mitochondrial dysfunction, respectively—posited mechanisms of action in PD—as well as symptoms in rodents similar to human PD. Now, researchers have linked human exposure to paraquat and rotenone with PD [***EHP* 119(6):866–872; Tanner et al.**]. Their study is the first analysis of pesticides classified by presumed mechanism of action rather than by intended use or chemical class.

The researchers assessed lifetime use of pesticides as reported by participants in the Agricultural Health Study, a prospective study of private pesticide applicators (mostly farmers) and their spouses in Iowa and North Carolina. In the nested case–control study, neurologists specializing in movement disorders identified 110 people with PD and 358 controls without who were frequency matched by age, sex, and state.

The researchers collected detailed information about use of 31 different pesticides as well as covariate information including lifelong smoking and family history of PD. Each pesticide was classified according to its mechanism of action as either an oxidative stressor or a mitochondrial complex I inhibitor. For participants with PD, the researchers considered pesticide use that occurred before their PD diagnosis. For controls, they considered pesticide use that occurred before the median age of PD diagnosis for cases in the corresponding age-, sex-, and state-specific groups.

Among the mitochondrial complex I inhibitors studied, the researchers found the strongest association between PD and use of rotenone. Among oxidative stressors, they found the strongest association between PD and use of paraquat. Participants with PD were 2.5 times more likely than controls to have reported use of rotenone or paraquat.

The results may have far-reaching implications, considering the widespread use of these pesticides. Paraquat remains one of the most widely used herbicides worldwide, and rotenone was used ubiquitously before most uses were voluntarily stopped in the United States in 2007. Other agents associated with mitochondrial complex I inhibition, such as permethrin, remain in common use.

The authors point out several limitations of the study, including the fact that participants reported exposure to many pesticides, and effects of other agents can't be excluded. However, the associations between PD and paraquat and rotenone remained after adjustment for overall pesticide use. It also was impossible to measure pesticide exposure, only estimate it retrospectively. Future investigations of combinations of pesticides and of other mechanistic groups will be important, as will mechanistic studies that model exposure conditions similar to those occurring in humans.

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Olympic Win

Lower Estimated Cancer Risk with Air Pollution Controls during the 2008 Beijing Games

Polycyclic aromatic hydrocarbons (PAHs) sorbed to fine atmospheric particulate matter ($PM_{2.5}$) increase inhalation cancer risk in exposed populations. In China each year, an estimated 6.5 people per million develop lung cancer due to PAH inhalation. But stringent air pollution control measures instituted during the 2008 Olympic Games in Beijing, if sustained over a lifetime, could reduce residents' PAH-related inhalation cancer risk by nearly half [***EHP* 119(6):815–820; Jia et al.**].

Coal combustion and motor vehicle emissions create severe air pollution in Beijing and other major Chinese cities. To improve air quality for the Olympic Games, factories in and around Beijing were moved or closed, vehicular traffic was restricted, and truck traffic was reduced during the period 20 July–20 September 2008. During the games themselves (8–24 August), even stricter controls were imposed.

$PM_{2.5}$ samples were collected at a single representative location in Beijing, and associated concentrations of 17 PAHs were measured and compared for four periods: 1) 28 July–20 September versus 2) 21 September–7 October, and 3) 8–24 August versus 4) 28 July–7 August/25 August–7 October. To enable direct comparison of chemical risks, a benzo[*a*]pyrene equivalent (BaP_{eq}) concentration was estimated for each PAH by multiplying its concentration by its relative potency factor (RPF). Cancer risk was calculated using the BaP_{eq} values paired with previously established unit risk measures for cancer based on a lifetime (70 years) of BaP exposure at 1 μg/m^3 air.

Individual BaP_{eq} concentrations were 22–78% lower in period 1 compared with period 2 (i.e., when any pollution controls were in place) and 32–72% lower in period 3 compared with period 4 (i.e., when the strictest controls were in place). Lifetime excess inhalation cancer cases estimated during the period when pollution was controlled ranged from 6.5 to 518 individuals per million compared with a range of 12.2–964 per million after pollution controls ceased—a 46% reduction. PAHs from the U.S. Environmental Protection Agency's list of priority pollutants made up three of the four largest contributors to total carcinogenicity; however, high-molecular-weight PAHs—a highly carcinogenic group of chemicals that, as a class, haven't been extensively studied—contributed a considerable 23% of the cancer risk. These top four PAHs are primarily associated with $PM_{2.5}$, so reducing $PM_{2.5}$ emissions would also reduce levels of these and other PAHs.

Limitations of the study include potential inaccuracies due to its point-estimate approach, which assumes additive cancer risk, and the RPF values, which were based on toxicologic studies with their own uncertainties. However, the study strongly supports the effectiveness of air pollution source control measures and also demonstrated the need to include high-molecular-weight PAHs in future studies.

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Early morning rush hour, Tiananmen Square, Beijing